Early Embolization for Ruptured Aneurysm in Acute Stage of Subarachnoid Hemorrhage with Neurogenic Pulmonary Edema

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Key words: pulmonary edema, endovascular treatment, spinal drainage, subarachnoid hemorrhage

Summary

Four cases of ruptured aneurysmal subarachnoid hemorrhage (SAH) presented with severe neurogenic pulmonary edema (NPE). On admission, two patients were grade IV and two were grade V according to Hunt and Hess grading. All patients needed respiratory management with the assistance of a ventilator. Three of them underwent endovascular treatment for the ruptured aneurysms within three days from onset after ensuring hemodynamic stability. Immediately after the endovascular treatment, lumbar spinal drainage was inserted in all the patients. The pulmonary edema findings disappeared rapidly after the respiratory management. The results were good recovery in two, and moderate disability in two. We concluded that early embolization of ruptured aneurysm and placement of spinal drainage is a satisfactory option for severe SAH with NPE.

Introduction

It is well known that neurogenic pulmonary edema (NPE) may develop acutely in patients with sustained severe subarachnoid hemorrhage (SAH) and may worsen the patient's general medical condition. Recently, embolization with detachable coil has been recognized as a less invasive treatment for cerebral aneurysm. We report herein four patients of severe aneurysmal SAH with NPE who were successfully treated early by embolization of aneurysm.

Patients and Methods

Between April 2002 and May 2005, four patients (one male and three female) with aneurysmal SAH with NPE were treated by endovascular treatment within three days of ictus. Patients ranged in age from 31 to 62 years (mean 50.3 years). Two patients presented with Hunt and Hess grade IV and the remainder with Hunt and Hess grade V. All had signs of acute respiratory failure with foamy sputum. Diagnosis of SAH was based on computed tomography scan. NPE was defined as the presence of all of the following criteria.

1. Immediate onset of Respiratory failure after ictus.
2. Chest X-ray film on admission revealed interstitial edema in bilateral lung fields.
3. No history of cardiac and lung disease.

Results

The four patients are summarized in the table. All patients required early intratracheal intubation and successfully underwent endovascular treatment following diagnostic angiography. After the endovascular treatment, continuous lumbar spinal drainage was placed for early discharge of SAH and control of intracranial

pressure. All required respiratory management with a mechanical ventilator, and the pulmonary edema in all patients disappeared within six days. Two of the patients achieved good recovery, but two patients suffered mild disability due to the symptomatic vasospasm.

Representative Case

Case 2

A 55-year-old woman with a history of hypertension was admitted to our hospital with sudden onset of unconsciousness. She presented with a decerebrated posture and respiratory dysfunction with pinkish foamy sputum on admission. A computed tomography scan showed diffuse subarachnoid hemorrhage, and a chest rentogenogram revealed diffuse pulmonary infiltrates in the bilateral lung field indicating the presence of pulmonary edema (figure 1). She was intubated immediately after admission and was controlled by continuous positive pressure ventilation with positive end-expiratory pressure. An angiogram was performed under general anesthesia after 15 hours of onset. Right carotid angiogram revealed a saccular aneurysm at the right middle cerebral artery (figure 2A). The patient underwent endovascular embolization with Guglielmi detachable coils (GDCs, Boston scientific Co.) following the diagnostic angiography. Complete occlusion of the aneurysm was performed (figure 2B). Immediately after the embolization, continuous lumbar spinal drainage was placed for one week.

Neurological recovery was incomplete, and mild aphasia was present due to the symptomatic vasospasm. Respiratory recovery was good, and the endotracheal tube could be removed on the sixth day; serial chest roentgenogram revealed complete resolution on the seventh day.

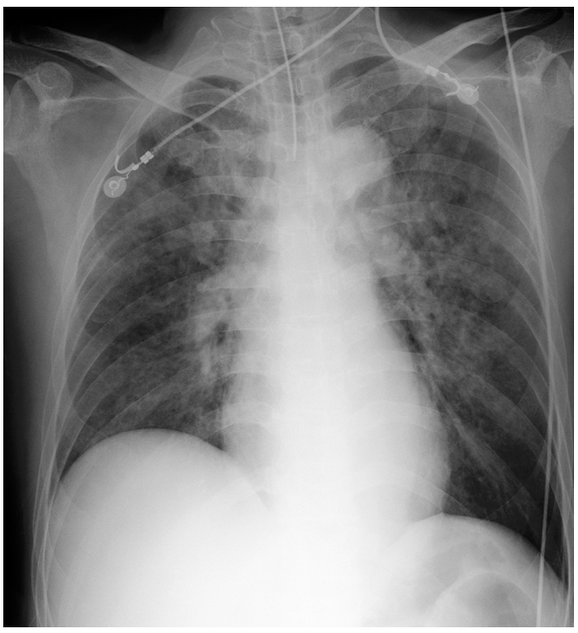

Figure 1 Chest X-ray shows diffuse pulmonary infiltrates in the lung indicating the presence of pulmonary edema.

Discussion

Weir has reported that an incidence of NPE complicating fetal SAH of 31% on the basis of clinical criteria and up to 71% in autopsy-reviewed cases [1]. It has been previously described that NPE most often develops within minutes to a few hours after an acute central nervous system [2,3]. There have been few reports regarding the best management for subarachnoid hemorrhage with NPE in the acute stage. Although early surgical intervention and aggressive management of delayed vasospasm are now standard procedures in the treatment of patients with aneurysmal SAH, there is a therapeutic dilemma between the policy of early aneurysm surgery and the successful management of pulmonary edema during radical intervention. Yabumoto et Al [4] have insisted on early and aggressive management of the ruptured aneurysm and symptomatic treatment of NPE. They have proposed that the syndrome of NPE should not be an obstacle to radical intervention when cardiorespiratory control can maintain the minimal anesthetic limit.

Recently, endovascular treatment has been recognized as a less invasive treatment for cerebral aneurysm than direct surgical clipping [5]. The most important advantages of endovascular treatment for aneurysmal SAH is that it can immediately follow the diagnostic angiogram and that the period required for intervention is shorter than direct clipping. We therefore suggest that endovascular treatment should be a first choice for poor-grade aneurysmal SAH with NPE.

NPE should be treated symptomatically, and early intubation is recommended. The mechanism by which NPE occurs is unclear. Several experimental investigations suggest that a sudden increase in intracranial pressure or hypo-

Figure 2 A) Anterior posterior view of right internal carotid injection reveals a right middle cerebral artery aneurysm. B) The aneurysm was embolized using GDCs.

thalamic lesions may elicit massive sympathetic discharges, with redistribution of blood to the pulmonary circulation resulting in high pulmonary capillary pressure and increased permeability [6,7]. Therefore, it is beneficial that treatment of NPE be used to control intracranial pressure and reduce subarachnoid hemorrahge by early placement of continuous lumbar spinal drainage. Patients who experience NPE after aneurysmal SAH have a higher incidence of symptomatic vasospasm than do patients without NPE [8]. This is most likely a result of conservative fluid management and a use of diuretics to promote adequate oxygenation in these patients. Early placement of lumbar spinal drainage also has a favorable impact on the development of delayed vasospasm.

Conclusions

We suggest that early embolization of aneurysm and placement of spinal drainage are an excellent treatment option for severe SAH with NPE.

Table 1 **Summary of Cases.**

Case No.	Age/Sex	H & H Grade	Aneurysm location	Interval between SAH and surgery	Surgical procedure	Period of tracheal intubation	Clinical outcome
1	31/M	IV	PCoA	2 h	Aneurysm embolization	6 days	GR
2	55/F	IV	MCA	15 h	Aneurysm embolization	6 days	MD
3	53/F	V	BA-AICA	6 h	Aneurysm embolization	24 hrs	GR
4	62/F	V	IC-PC	68 h	Aneurysm embolization	5 days	MD

PCoA; posterior communicating artery, MCA; middle cerebral artery, BA-AICA; basilar artery - anterior inferior cerebellar artery IC-PC; internal carotid artery - posterior communicating artery, GR; good recovery, MD; moderately disabled

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